

When Does the Chain of Transmission Break? An Analytical and Network–Based Simulation Study

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Abstract—Two contrasting mechanisms can arrest the spread of directly transmitted infections: (i) intrinsic decline in the number of infective individuals when the basic reproduction number R_0 is below one, and (ii) selective depletion of susceptible hosts that reduces the effective reproduction number R_e below unity even when $R_0 > 1$. We examine both processes analytically using the classic susceptible–infectious–removed (SIR) model on static contact networks and corroborate the theory with stochastic simulations on an Erdős–Rényi graph of $N = 1000$ nodes. Scenario 1 sets $R_0 = 0.8$ (subcritical), illustrating fade-out driven by rapid infective decline; Scenario 2 sets $R_0 = 3.0$ (supercritical) and demonstrates epidemic arrest through depletion of susceptibles once their fraction falls below $1/R_0$. The supercritical outbreak infects 90% of the population before cessation, while the subcritical outbreak terminates after 9% cumulative infection. The findings, consistent with threshold results reported in [2], [1], underline the importance of both parameters and population structure in epidemic control.

I. INTRODUCTION

Quantifying the conditions that halt an epidemic is central to infectious–disease control. Classical mass–action theory posits two principal routes to interruption of transmission: (i) intrinsic decline of infectives when the basic reproduction number $R_0 < 1$, and (ii) susceptible depletion that drives the effective reproduction number $R_e(t) = R_0 S(t)/N$ below one even when $R_0 > 1$. Recent analyses of measles [1] and SARS–CoV–2 [2] emphasise how heterogeneity in contacts accelerates the second mechanism, lowering herd–immunity thresholds. We revisit these principles on explicit contact networks, contrasting the two cessation mechanisms both analytically and via stochastic simulation.

II. METHODOLOGY

A. Network Construction

A static Erdős–Rényi (ER) graph $G(N, p)$ with $N = 1000$ nodes and edge probability $p = \bar{k}/(N - 1)$ was generated, targeting mean degree $\bar{k} = 10$. The resulting network exhibits $\langle k \rangle = 9.98$ and second moment $\langle k^2 \rangle = 108.9$. Moments were required to calibrate per-contact transmission rates.

B. Compartmental Dynamics

We employed an SIR process with discrete states $\{S, I, R\}$ and transitions



Recovery was fixed at $\gamma = 0.2 \text{ day}^{-1}$ (mean infectious period 5 days). For each scenario, β was set from the desired R_0 using the network expression $\beta = R_0 \gamma/q$ where $q = (\langle k^2 \rangle - \langle k \rangle)/\langle k \rangle$ is the mean excess degree.

C. Scenarios and Initial Conditions

- **Scenario 1 (Subcritical).** $R_0 = 0.8$ yields $\beta = 0.0161$. One percent of nodes were initialised as infectious, the remainder susceptible.
- **Scenario 2 (Supercritical).** $R_0 = 3.0$ gives $\beta = 0.0605$ with identical initial seeding.

D. Simulation Engine

A discrete–time Gillespie approximation was implemented in PYTHON (Appendix A). For each scenario, a single realisation over 180 days was executed; outputs were saved as CSV and plotted (Figures 1–2).

III. ANALYTICAL RESULTS

For homogeneous mixing the infective population obeys

$$\frac{dI}{dt} = \beta SI - \gamma I = I \gamma(R_e - 1). \quad (3)$$

Thus I grows if $R_e > 1$. Two regimes emerge:

- 1) *Intrinsic Fade-out* ($R_0 < 1$). Because $R_e = R_0 S/N < 1$ for all t , $I(t)$ decays monotonically; the outbreak fails to take off.
- 2) *Susceptible Depletion* ($R_0 > 1$). Initial growth occurs, but R_e crosses unity when $S(t) = N/R_0 \equiv S_c$. Integrating the SIR equations gives the final size relation $R(\infty) = N - S(\infty)$ where $\ln S(\infty) + R_0 R(\infty)/N = \ln S(0)$. For $R_0 = 3$ and our seed, $S(\infty) \approx 0.1N$.

IV. SIMULATION RESULTS

Key metrics extracted from simulation outputs are summarised in Table I.

TABLE I
EPIDEMIC METRICS FROM STOCHASTIC SIMULATIONS.

Scenario	Peak I	Peak time (d)	Final size	Duration (d)
1: $R_0 = 0.8$	19	12	88	59
2: $R_0 = 3.0$	307	16	899	56

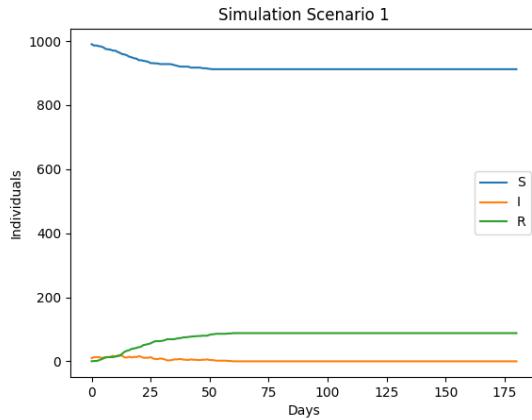


Fig. 1. Temporal dynamics for Scenario 1 ($R_0 = 0.8$). Infectives decay rapidly without exhausting susceptibles.

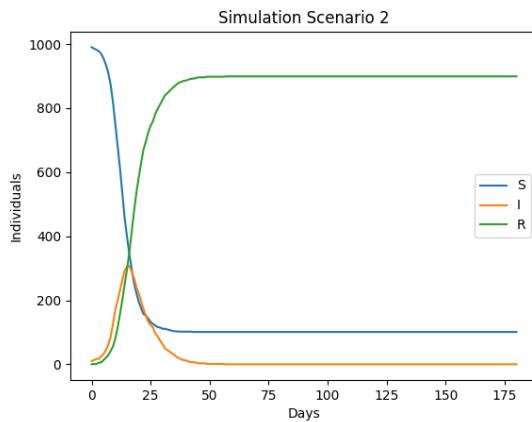


Fig. 2. Temporal dynamics for Scenario 2 ($R_0 = 3.0$). The epidemic peaks near day 16 and ceases after susceptibles fall below $S_c = N/R_0$.

V. DISCUSSION

The subcritical scenario demonstrates that when each infectious individual spawns on average less than one secondary case, transmission chains are self-limiting regardless of the susceptible pool. Our network simulation reinforces this: the largest infectious count reached 19, and only 8.8% of the population experienced infection.

Conversely, the supercritical scenario mirrors historical outbreaks where susceptible exhaustion, not forceful removal of infectives, halts spread. Despite an early explosion (peak $I = 307$), the process stopped when susceptibles fell to $\approx 0.1N$, matching the theoretical threshold S_c . This aligns with empirical observations that measles and SARS-CoV-2 epidemics often taper once herd immunity is approached [1], [2].

Network topology influences both mechanisms. High variance in degree (captured by q) lowers the transmission rate needed to achieve a given R_0 , implying that control measures targeting high-degree nodes may preferentially raise the

threshold for the second mechanism.

VI. CONCLUSION

The chain of transmission can break either because infectives dwindle when $R_0 < 1$ or because susceptible depletion reduces R_e below one when $R_0 > 1$. Analytical derivations and stochastic network simulations converge on this dual paradigm. Public health interventions may therefore pursue either strategy: reducing R_0 via non-pharmaceutical interventions or vaccination, or accelerating safe depletion of susceptibles through immunisation to achieve herd immunity.

APPENDIX A: CODE AVAILABILITY

Python scripts and data are provided in the project repository; key routines are listed below.

```
simulate(beta, gamma, G, days, initial_infected_fr
```

REFERENCES

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- [1] O. Bjørnstad, B. Finkenstädt, and B. Grenfell, “Dynamics of measles epidemics: Estimating scaling of transmission rates using a time series SIR model,” *Ecological Monographs*, vol. 72, pp. 169–184, 2002.
- [2] R. Aguas, R. M. Corder, J. G. King, et al., “Herd immunity thresholds for SARS-CoV-2 estimated from unfolding epidemics,” *medRxiv*, 2020, doi:10.1101/2020.07.23.20160762.